

## RELATIONSHIP AMONG SERIAL CORONARY ARTERIAL REMODELING, STENOSIS PROGRESSION, AND PLAQUE COMPOSITION: A VH-IVUS TISSUE CHARACTERIZATION ANALYSIS

i2 Poster Contributions

Georgia World Congress Center, Hall B5

Monday, March 15, 2010, 9:30 a.m.-10:30 a.m.

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Session Title: Intravascular Diagnostics and Complex Lesions

Abstract Category: Intravascular Diagnostics

Presentation Number: 2503-506

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**Background:** We studied the relationship among serial remodeling, plaque composition, lesion phenotype, and stenosis progression.

**Methods:** Of 990 pts who were enrolled in a prospective, 42 center, nonrandomized, global virtual histology intravascular ultrasound (VH-IVUS) registry, we identified 99 pts with serial (baseline and follow-up @  $11 \pm 5$  mos) VH-IVUS with 224 non-culprit, untreated lesions (plaque burden  $\geq 30\%$  in at least 3 consecutive frames). The 4 VH-IVUS plaque components were color-coded as dense calcium (DC), necrotic core (NC), fibrofatty (FF), and fibrotic tissue (FT) and reported as percentages of plaque area and total plaque volume. We classified lesions into 5 phenotypes: pathological intimal thickening (PIT), thin cap fibroatheroma (TCFA), Thick cap fibroatheroma (ThFA), Fibrotic (F), Fibrocalcific (FC).

**Results:** At the minimum luminal site, remodeling ( $\Delta$ EEM) correlated with  $\Delta$ lumen ( $p < 0.001$ ),  $\Delta$ plaque overall ( $p < 0.001$ ), and  $\Delta$ FF ( $p < 0.001$ ) and  $\Delta$ FT ( $p = 0.001$ ), but not with  $\Delta$ NC ( $p = 0.321$ ) or  $\Delta$ DC ( $p = 0.417$ ). When assessed by lesion subtypes, plaque increased significantly in PIT ( $p < 0.001$ ), TCFA ( $p = 0.016$ ), and ThCFA ( $p < 0.001$ ), but not in fibrotic or fibrocalcific plaque. Lumen area decreased significantly in PIT ( $p < 0.001$ ), TCFA ( $p = 0.006$ ), and ThCFA ( $p < 0.001$ ), but not in fibrotic or fibrocalcific plaque. However, EEM area did not change significantly from index to follow-up in any lesion subtype; and  $\Delta$ EEM was similar among the 5 subtypes:  $0.1 \pm 1.3$  mm<sup>2</sup> in PIT,  $-0.2 \pm 0.8$  mm<sup>2</sup> in TCFA,  $0.1 \pm 1.1$  mm<sup>2</sup> in ThFA,  $-0.1 \pm 1.5$  mm<sup>2</sup> in F,  $-0.2 \pm 1.0$  mm<sup>2</sup> in FC ( $p = 0.623$ ). Finally, there was no significant correlation between  $\Delta$ EEM and baseline plaque composition.

**Conclusions:** Serial remodeling correlates with plaque progression and influences stenosis progression (lumen compromise), but is not related to VH-IVUS lesion phenotype or to the change in necrotic core. However, VH-IVUS phenotypes do predict stenosis stability independent of serial remodeling; PIT, TCFA, and ThCFA are associated with plaque progression and increasing lumen compromise while fibrotic and fibrocalcific plaques are stable and do not progress.